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Held in Washington, D.C.

WORLD TRENDS IN CARDIOLOGY

I Cardiovascular Epidemiology
II Cardiovascular Surgery
III- Blood Volume and Contractile Protein in Heart Muscle
IV. Cardiovascular Diagnosis and Therapy
V. Instrumental Methods in Cardiac Diagnosis

World Trends In Cardiology: II

Cardiovascular Surgery

PANEL DISCUSSIONS

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FOREWORD

It is always profitable and interesting to study the opinions of recognized experts and to know the foundations for their particular views. Usually, as we read, we have questions which must go unanswered until we encounter the expert face to face or a subsequent publication provides the answers. Often, we would like to ask how his view holds against the dissimilar or perhaps contrary view of another expert, or how new or collateral developments might affect his view. Seldom is an on-the-spot opportunity provided for such questioning.

Here, from the panel discussions on cardiovascular surgery at the Second World Congress of Cardiology, are recorded the results of such an opportunity. Not one but several experts in each of the surgical fields under discussion have expressed their previously considered views on the subjects in question and then, in extemporaneous fashion and with such facts as they could at the moment draw from their own experience, weighed their views against those of their colleagues. The result is an exciting and authoritative look into the dynamic and spectacular field of cardiovascular surgery and one which, through the stimulating questions of the moderators and the answers of the other experts, provides much insight and food for scientific thought.

H. B. T.
A. S. C

CONTENTS

Panel I

Medical Aspects of the Surgical Treatment of Congenital Heart Disease

PATENT DUCTUS ARTERIOSUS

CHAIRMAN HELEN B. TAUSSIG: The diagnosis of a patent ductus arteriosus is usually based on the presence of a continuous murmur over the pulmonary area, in a noncyanotic patient. I *am going to ask each of these panelists to indicate* how many patients have been operated on in whom the diagnosis was based on these simple criteria, the errors in diagnosis, and the percentage of error. Dr. Campbell, will you give us the English experience?

DR. MAURICE CAMPBELL: I don't think I have submitted any patient for operation with an incorrect diagnosis, out of about 100 patients. I went through them carefully, and I thought there were about 5 per cent where one could have real clinical doubt, and whenever I have any, patients are submitted to catheterization. Dr. Soulie of France has told us how reliable catheterization is in settling that; so I feel that my failures in diagnosis have been in the other direction, in missing the case that was a patent ductus arterious although it did not have the classical signs of it.

I have been distressed at 2 deaths from bacterial endocarditis in small children who were awaiting admission for investigation by catheterization. I did not think they were cases of patent ductus arterious, but they were.

CHAIRMAN TAUSSIG: Dr. Durand, what has been the F experience?

DR. M. DURAND. In 101 cases submitted to surgery, we 4 errors in diagnosis. The first was an atrial septal defec girl 2 years old. In the second case, there was an arterial and an arteriovenous fistula. As yet we are not certain abo second case, because surgery has not been completed. arterial canal was operated upon, and it was rather small. the operation, the murmur continued at the base of the so we believe that there is an arteriovenous fistula.

The third error occurred in a case of coarctation of the of a rather special type, in a child 6½ years old. The c femoral pulsations were quite normal. There was no ar canal and there was stenosis of the aorta in the region adj to the arterial ligament. The stenosis was not great, and permitted circulation. But auscultation at this level revea continuous murmur over the sternum. This is what we hear thought was the arterial canal.

The fourth error was made in a girl 4 years old. We fo during the operation, that there was no arterial canal, but there was an arterial trunk with a left branch, coming di from the aorta. There was no pulmonary artery.

CHAIRMAN TAUSSIG. Thank you, I asked for the inciden errors to bring out the relative safety of the diagnosis wh is based upon a continuous murmur within the pulmonary Dr Kreutzer, may we please have the Latin American ex ence?

DR RODOLFO O. KREUTZER: Of 109 patients who were ated on with a diagnosis of patent ductus arteriosus, 94, or per cent, had the essential diagnostic feature: a contin murmur. In these 94 patients, patent ductus arteriosus was fo and ligated with success in 87, or 92 6 per cent. In 4 of the c or 4 95 per cent, the ductus was found, but the continuous mur did not disappear, either during the clamping or afte ligation of the ductus arteriosus. These patients suffered rela after this operation. In 1 case which was reoperated upon,

ductus was found to be completely occluded In spite of this, the patient has suffered relapses with enlargement of the heart.

In 3 cases, or 3 5 per cent, the ductus was not found during the operation, in spite of a continuous murmur. All 3 of these patients died shortly after operation, and in none of them was it possible to obtain an autopsy. In 1, there was probably an aortic septal defect with bacterial endocarditis. In the others, we have a diagnostic error which cannot be cleared up.

CHAIRMAN TAUSSIG· Dr. Gibson, will you tell us what has happened in America.

DR. STANLEY GIBSON: Our report from the 15 cardiac centers in this country indicated 3433 operations. In 2252, the ductus was divided; in 1181, the ductus was ligated The total mortality was 68, or 2 per cent.

The errors in diagnosis, based upon the presence of a characteristic continuous murmur at the base of the heart, were 30, or only 1 per cent. This did not, of course, include atypical cases, in which the characteristic continuous murmur was absent; so I think these figures show that diagnosis can be made fairly accurately with a stethoscope, and that the operation is, in good hands, a very safe one.

CHAIRMAN TAUSSIG: Dr. Snellen, will you comment?

DR H. A. SNELLEN: I think the main question was whether or not a continuous murmur was a safe criterion for diagnosis. It appears that it is, and that, perhaps, the only condition which causes difficulty is the aortic septal defect, which is rare and not easily recognized even with more elaborate procedures like catheterization of the heart.

The other conditions—such as arteriovenous fistula and the venous stenosis of the innominate vein—may be easily recognized in most cases. Therefore I think we can rely on the continuous murmur, and I think that is the main point in this discussion.

CHAIRMAN TAUSSIG: It is now nearly fifteen years since Dr. Gross operated on his first patent ductus arteriosus, and in the last ten years the operation has been widely done throughout this country and Europe. Indeed, today we have had a report

not very much overriding aorta, and severe disability, he might not be suitable for an anastomotic operation and might be much more suitable for a direct one. In fact, in the early stages, we were frightened about doing anastomotic operations in some of those patients, but the results of doing them were just as good, and I don't think one can make that a criterion.

At one time, because of the risk of increasing heart size after the extra work of an anastomosis, it seemed that the patients might be particularly suitable for direct operation where the heart was already larger than usual. I do not think this is so, because our recent follow-up shows that the tendency of the heart to increase is exactly the same in both postoperative groups. It is not very great, but it is present in both of them, and it seems to me that it must be an inevitable risk in any operation that improves the condition of these patients until surgery advances and their ventricular septal defect can be closed as well.

I think one must admit that the direct operations carry a greater operative risk, but the operative risk of anastomotic operations has diminished during the last few years. My own experience is an 8 per cent mortality with the anastomotic operations, an 11 per cent mortality with valvulotomy alone, and a 15 per cent mortality where there has been infundibular section alone or with valvulotomy.

The good results are extraordinarily similar, and I need not go into them in detail because we have just published a paper comparing the results. It is almost uncanny, the agreement in the figures between those classified in the very good and the good groups, and the average heart size after one to four years. I think, really, that might have been anticipated.

In my opinion the greatest contribution of Dr. Taussig was her appreciation of this problem. Discussion had been going on for several hundreds of years on the importance of diminished blood flow to the lungs. Both these operations increase the blood flow to the lungs, both of them leave ventricular septal defects, and one might expect the results to be the same.

Finally, on the late follow-ups, I am not quite as optimistic

about the lasting results as I was when we last published a follow-up of anastomotic operations. The fourth and fifth years have begun to show a few patients who are losing ground. Some are getting closed anastomoses, and some are getting increasing right ventricular strain. This is merely a guess, based on fairly intelligent knowledge of a large number of patients who have been operated on. I feel that, perhaps, ten or fifteen years will be the period of improvement we can expect for both these operations, until surgery has advanced enough to close the ventricular septal defect as well

These figures are based on 200 anastomotic operations. Four per cent have died or developed severe failure. Another 2 per cent have had second operations for failure to maintain improvement. That is, 6 per cent in all.

The direct operations number 100. The number who have relapsed is smaller, but then so is the period of follow-up. Their period of follow-up is about the same as those reported in our first publication on the anastomoses and there only 2 per cent have deteriorated. But there are 4 per cent who have needed a second operation or a different type of operation because the results were not satisfactory.

DR. DURAND: Our surgeons have operated on 420 patients for tetralogy of Fallot since 1947, and only 19 were done directly, which represents 95.7 per cent for the anastomoses In the great majority of cases, these were of the intrinsic type. The mortality in these 420 cases was 68, or 5 1 per cent. The good results, in the two months after operation, totaled 74 per cent of the cases.

Nineteen were undertaken by direct attack, of which 4 were by ventricular valvulotomy, 4 by infundibular valvulotomy, and 4 by a retrograde arterial manner. Five of the 19 died during the operation or a few hours thereafter. Ten out of the remaining 14 survived.

There have been follow-up studies on 57 of the patients who had anastomotic proceedures. Forty showed good results five years later, and 5 had a second operation because of failure to maintain improvement.

It seems that after a period of adaptation, there is stabiliza-

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mortality has been 3 per cent. Errors of diagnosis were reported as 119 out of the total group, but figures are often deceiving

Good results were reported in 70 per cent. Severe heart failure was reported in 69 patients or 2 6 per cent. A total of 99 second operations had to be done; in 3 7 per cent of the entire group. In many instances, the anastomosis had closed, and I think that happened most often in the younger patients

I might mention that subacute bacterial endocarditis occurred 15 times in the early postoperative period; 16 times in the late postoperative period. Of course, that does not represent the total incidence because of our inability to follow all patients closely for a long period of time.

DR. SNELLEN: Dr. Campbell has brought out the fact that the results are about the same following the direct attack or the shunt operation, but the danger is certainly less with the shunt. That is why we prefer the shunt operation

CHAIRMAN TAUSSIG: I am sure that our American mortality rate has been considerably higher with the direct attack than with the shunt operation. I think it remarkable that our errors of diagnosis have been so low. The mortality rate has varied, as Dr. Gibson said, from clinic to clinic depending on the groups It has been as low as 3 per cent in one very special group and as high as 20 in another. The mortality rate in our group is high, I must confess, but Dr. Blalock is generous and operates on any patient whom I think will be improved if the patient can survive the operation. We feel that the difficult patient should be referred to us, and we are willing to take the risk, even though it means our figures will not be as good as others

Almost all of us have come out the same, with good results in more than 70 per cent Seventy per cent of those who obtained good results have maintained their good results.

COARCTATION OF THE AORTA

CHAIRMAN TAUSSIG: Next we will ask our panelists whether or not they think all patients with coarctation of the aorta should be operated on. If not, will they give us three or four of the essential criteria and the contraindications for operation.

DR. CAMPBELL: I certainly would not agree that every case of coarctation should be operated on. We need a great many more years of follow-up to see how they do before one can reach that conclusion.

My criteria for operation are: (1) increasingly severe symptoms; (2) rising blood pressure, and (3) indications of potential cerebral hemorrhage.

The contraindications are: (1) no symptoms and no enlargement of the heart in a patient who seems to be getting on perfectly well; (2) disability that is due primarily to gross aortic incompetence; and (3) demonstration by special investigation that the coarctation is not a narrow, simple constriction and will need a graft. I think we must accept the fact that use of a graft greatly increases the operative risk.

DR. DURAND: The major indications for operation on coarctation are: (1) the amount of hypertension; (2) cerebral difficulties such as syncope, impaired speech, or embolic phenomena; or (3) cardiac enlargement with left ventricular hypertrophy or evidences of progressive increase in size during period of observations. If one has a choice—that is, if one sees the person when he is only 8 or 10 years old—present evidence indicates it is better to wait and operate when he is between the ages of 12 and 16. At the later age, the surgical condition is still good, but the patient is old enough for somatic development to be established.

The contraindications are, first, the age. It is obvious that, if the patient is over 30 years old, and the coarctation is well tolerated, it is better not to operate. Second, signs of heart failure seen clinically or radiologically, or other symptoms of hypertrophy with disturbance of conduction, must be considered contraindications.

DR. KREUTZER: We do not believe that every patient with coarctation of the aorta should be operated upon. The principal criteria for operation are. (1) the patient is at least 5 years of age or older; (2) a fixed hypertension of the upper extremities, with almost negligible femoral pulse; or (3) there should be cardiac enlargement.

We consider the following to be contraindications: (1) age below two years, (2) clinical or radiologic evidence that suggests hypoplasia of the aorta, or subendocardial fibrosis with or without mitral stenosis.

DR. GIBSON. Figures have been gathered from 15 cardiac centers throughout our country. The total number of operations has been 1279. The mortality has been 57 or 4 5 per cent. Bacterial endocarditis has been relatively rare. There were 5 cases in the early postoperative period, and 2 cases in the late postoperative period. Obviously, the indications and the contraindications for operation vary somewhat from clinic to clinic and I could not summarize all of this in a brief statement.

DR. SNELLEN: It is obvious that the consensus is to wait and to see if anything serious develops, such as increasing hypertension or early evidence of cerebral disturbances, before operating.

In my opinion, indications for operation depend largely on the surgical technic and the safety of the procedure In general we feel that we should recommend operation at an early age because of the severity of later symptoms, regardless of hypertension. The main contraindication is heart failure.

CHAIRMAN TAUSSIG. What has been the incidence of subacute bacterial endocarditis? It is ten years since this operation started, and we should have some indication whether the correction of the malformation reduces the incidence of subacute bacterial endocarditis. Therefore, I ask each panelist to give the size of the group and the number of cases of subacute bacterial endocarditis he has seen in the early postoperative period, and in the late postoperative period

DR. CAMPBELL: The numbers are small, because, until the last two or three years, Sir Russell Brock was fully occupied with the cyanotic congenital cases and coarctations could wait longer. Nevertheless, I have not seen postoperative bacterial endocarditis in any of the patients he has operated.

DR. DURAND: We know of no subacute bacterial endocarditis in the immediate or later postoperative period among 65 patients.

DR. KREUTZER: In Argentina the number of our cases operated on for coarctation of the aorta is very small. I have been able to gather 24 cases. None of them had any signs of subacute bacterial endocarditis, in either the immediate or the late post-operative period.

DR. GIBSON: Among 1279 operations, bacterial endocarditis in the early postoperative period was reported in 5 instances, and late bacterial endocarditis in only 2 instances, making a total of only 7 instances.

DR. SNELLEN: I know of 120 cases without a single subacute bacterial endocarditis. I think the whole situation is very gratifying, and that the danger, which is still slight, should be expected in the immediate postoperative period.

PURE PULMONARY STENOSIS

CHAIRMAN TAUSSIG: Our next question concerns pure pulmonary stenosis. I believe we all agree that pulmonary valvulotomy is the operation of choice in patients with pulmonary valvular stenosis and an intact ventricular septum. I should like to ask if they think that all patients with this type of pulmonary stenosis should be operated on, and, if not, the two or three outstanding indications for operation

DR. CAMPBELL: I feel even more strongly with pulmonary stenosis than with coarctation of the aorta that all cases should not be operated on. Our follow-up at Bristol in England has shown how well many of these cases do from childhood to middle age, and I have recently seen a patient of 66 who had had no trouble until the last year.

Those who pose the question assume the surgeon has miraculous powers to replace the stenosed valve by a normal one. We should remember that he is not yet able to do that. The surgeon is able to change a case of severe stenosis with gross symptoms into a case of trivial stenosis with no symptoms, with some risk of producing pulmonary regurgitation. Those risks make it very clear that patients should not be operated on unless they have symptoms.

My three main criteria for operations are: (1) considerable

enlargement of the heart, and I think that means that you have left it too late and added to the risk, (2) increasing signs of right ventricular strain, by electrocardiogram, which I regard as extremely important, and the best indication that the patient is going the wrong way; (3) we have tended to accept a right ventricular systolic pressure of 100 mm. or over as an indication for operation.

DR. DURAND: In the cases of pulmonary stenosis, only certain cases must be considered. Obviously, an operation is not at all indicated if there is no important functional disturbance nor indications on the electrocardiogram of right ventricular strain. Besides the radiologic and functional elements, one must avail oneself of the hemodynamic data and see what is the increase of pressure in the right ventricle and the difference between the right ventricle and the pulmonary artery, to determine the degree of stenosis. When these criteria do not tell us to operate, a good test is that of evolution, which will tell us whether to operate, in the years to come, on patients who eventually develop hypertrophy.

DR. KREUTZER: In the beginning of my career as a physician, the diagnosis of pulmonary stenosis was made with great frequency. Later, it was considered to be very rare. Today, with new research methods and the use of cardiac catheterization, once more pulmonary stenosis is regarded as a common congenital cardiopathy.

I do not think that all cases of pulmonary stenosis should be operated upon. Our experience supports Dr. Campbell: there are many patients who tolerate this cardiopathy and in whom stenosis is only discovered by routine cardiac catheterization. I believe that we should operate on those patients in whom: (1) there is an increase in the size of the heart with or without cardiac insufficiency, (2) the electrocardiogram shows hypertrophy of the right ventricle; and (3) the pressure of the right ventricle is over 120 mm. of mercury. I am not even completely sure that patients with pressure of 120 mm. of mercury should be operated on, because I have seen many who tolerate this hypertension quite well.

DR. GIBSON: Our total number of operations for pure pulmonary stenosis is 524; the mortality was 32, or 6 per cent. There were 3 instances of bacterial endocarditis, among the 524 patients.

The indications for operation were very interesting among this group of 15 different surgeons. The chief criterion seemed to be the height of the right ventricular pressure. Some operated when it is above 60 mm.; some, 70 mm.; some, 80 mm. It was practically unanimously agreed that if the right ventricular pressure was 100 mm. or more, operation should be done.

As to the other indications, I cannot list them here. I found some variation, I think, depending upon whether the surgeon was dealing chiefly with adults or children. The indication in children with early cardiac enlargement is emphasized, whereas in the adult the heart may not be so much enlarged.

DR. SNELLEN: I think there is agreement that we should not operate on all cases of pulmonary stenosis, and it is obvious that there are many persons who have no symptoms at all and who live to an old age with that lesion. On the other hand, I think we should know that there is a stage of pulmonary stenosis when cardiac insufficiency and heart failure supervene very quickly. We should avoid that, and a very good solution, which practically all of us have suggested, is to take a certain limit of pressure in the right ventricle—for instance 100 mm. of mercury—as a dividing line.

CHAIRMAN TAUSSIG: Thank you. Next, I should like to ask what the incidence of subacute bacterial endocarditis has been in the group with valvular pulmonary stenosis. I do not think any of us feel that the circulation has been restored to normal. The situation is different than in a case of a ductus arteriosus, which is ligated or divided. Here, as Dr. Campbell said, we have a valve which is still abnormal after it is cut; so I would not suppose that we have eliminated the risk of subacute endocarditis.

DR. CAMPBELL: My answer is again short. There have been no cases of bacterial endocarditis in 60 patients who have been operated on, during follow-up periods up to four and five years.

DR. DURAND: In the postoperative period we did not see a single case of subacute bacterial endocarditis in the 35 patients who were operated on.

DR. KREUTZER: Of 23 cases of pulmonary stenosis that have been operated on, the operative mortality was high. Seven died, approximately 30 per cent. No sign or suspicion of subacute bacterial endocarditis has been noted in the survivors.

DR. GIBSON: There were 3 cases among 524 children and those were all in the early postoperative period

I do not think this means that we should not give prophylactic antibiotics during the early postoperative period. None of us wish to suggest that. I am sure that we all advocate giving prophylactic antibiotics in large doses during the early postoperative period, until the wound has healed—between four days and two weeks.

Panel II

Surgical Treatment of Congenital Heart Disease

ATRIAL SEPTAL DEFECTS

CHAIRMAN ALFRED BLALOCK: Where should one draw the line in bringing patients with septal defects to surgery? The work of the heart is apt to be very high in some of the people with great left-right shunts through these openings, and there does not seem to be much doubt that the larger defects should be brought to surgery.

In general, we have felt—and this may be changed with future experience—that if the flow through the right ventricle is twice that through the left ventricle or higher, we should recommend surgery. We realize that that dividing line may be changed in the years ahead, as more experience is gained.

DR. ROBERT E. GROSS: We have learned already that there are two conditions which present formidable obstacles in these cases, and patients presenting them should not be brought to surgery.

The first of these conditions is a greatly increased pulmonary bed resistance. After there is a great flow to the pulmonary circuit for a good many years, the resistance in the vascular bed rises. If studies indicate that the resistance is high, the risk of surgery is very high. If the resistance has climbed to the point where blood is damming back in the right side of the heart and reversing the direction of the shunt, we believe this to be a

definite contraindication to operation. We have had 3 fatalities in such cases.

The second group with which we have had trouble includes patients with *very low defects of the ostium primum type.* Really, this situation is complicated. It is not simply a large hole situated rather low in the septum There is likely to be a disturbance of conduction as well, such as dissociation of auricular and ventricular beats, and deformities of the valves, in which the septal edges of the tricuspid and the mitral valves are deficient. There is thus a regurgitation through these two valves, which is just as important—perhaps more important—than the opening in the septum. We feel at the moment that these low defects with valvular anomalies represent a strong contraindication to the operation as we know it today.

DR. C. WALTON LILLEHEI: I agree with Dr. Gross on the septum primum defects with the closed technic. However, with the open, cross-circulation technic we have repaired atrial ventricular canals successfully, and I think these defects are more severe than the septum primum defects By that technic, conduction difficulties, which have been constituted the main problem in repairing those defects by other methods, are obviated.

DR. GROSS: I suppose each surgeon should adopt the method with which he is most familiar and which gives him the best results, realizing that there are other methods which in someone else's hands are just as good or perhaps better. We have not used any of the open technics, though there are, obviously, certain advantages in their use. We have, in about half of our cases, used an open well, which allows us to get into the heart in a blind manner but yet in a rather leisurely way, and close defects by direct silk suture or, if they are large, by the onlay of some plastic material to cover the defects. In the other cases, we have used an atrioseptopexy, inverting the lateral part of the posterolateral wall of the right auricle to plug the opening. I think this is a very effective operation under certain circumstances but it certainly is not applicable to all cases

SIR RUSSELL: I feel that one must stand back and look at this problem of closure of atrial septal defects with some de-

gree of perspective. I cast my mind back to the earlier days of the surgical treatment of bronchiectasis. Before it was possible to remove the diseased lung, there was a whole list of methods for treating it—postural drainage, artificial pneumothorax, phrenic avulsion, total pneumonectomy, thoracoplasty, pneumoperitonium, and so on. As soon as one-stage lobectomy became established, the other methods vanished almost overnight. Today, if you are going to treat a case of bronchiectasis surgically, you cut it out.

We are in the same position with intra-atrial septal defects. There are all kinds of neat methods which beat about the bush. You sew it up with various clever maneuvers from the exterior of the heart or with the use of a well. I think these methods are going to be of historical interest. If you have got a hole in the heart, the correct thing to do is to sew it up. So I feel that it is only a question of time until the open methods are just as firmly established as one-stage lobectomy for bronchiectasis.

CHAIRMAN BLALOCK: I believe that is the ideal, without doubt. On the other hand, I am sure there are enough surgeons who have had experience in this field to show that more simple methods have been very effective in many cases.

DR. C. WALTON LILLEHEI: I might relate the experience of Drs. Lewis and Farcot at the University Hospitals with the open technic and hypothermia. They have now operated on 19 intra-atrial septal defects in all ages, but mostly in the older age groups and groups in which this defect causes the most trouble. There have been 2 deaths. There has been 1 additional patient who fibrillated and the operation was not completed, although he survived the fibrillation. He has not yet returned for repair. All the rest have been succesful One patient still has a left-right shunt, apparently from anomalous drainage into the superior vena cava, which was not recognized. All of these were closed by direct suture.

CHAIRMAN BLALOCK: You have used no grafts of plastic materials?

DR. LILLEHEI: It is my feeling that one does not need them with the open technic. As a matter of fact, prostheses

are in many respects disadvantageous. They take more time to put in, they are abnormal, and they may cause trouble at a later date. With the heart open and relaxed, *there is no difficulty* in bringing the opposing edges together by the simplest method —direct suture.

CHAIRMAN BLALOCK: What proportion of these operations were done under hypothermia and what proportion under controlled cross-circulation?

DR LILLEHEI. We feel that hypothermia is the method of choice for simple intra-atrial defects because of its simplicity. It gives ample time to complete the operation. All of ours were done under hypothermia.

COARCTATION OF THE AORTA

CHAIRMAN BLALOCK: Dr. Mathey, what are your indications for surgery in infants with coarctation?

DR JEAN MATHEY: *We have been able to divide coarctations* into three groups In the first group, the signs of left ventricular hypertrophy are not marked and seem to be stabilized after the first examination. Under these conditions, we believe that surgery is not necessary during the first 2 years of life, and one can wait until the optimal age of 3 or 4 years. The future will tell whether it is better to operate as soon as the diagnosis is made or to wait for the third or fourth year.

On the other hand, the children who present signs of progressive left ventricular hypertrophy seem to be in danger. Indeed, all the children with these findings died between the ages of 3 months and 18 months. We therefore think that their prognosis is bad and surgery is indicated

If the signs of pulmonary congestion or bronchopulmonary difficulty are not very severe, the operation may be considered safe and beneficial On the other hand, in certain cases, pulmonary phenomena place a very different aspect on the question, because the bronchial defect and the atelectasis may make the operation very difficult. The problem under these conditions is difficult because in many cases, medical treatment cannot take care of the pulmonary phenomena. In one instance we waited

on medical therapy and the child died within a few weeks. We believe that in the future these patients might be treated in a more effective way through hypothermia, which would reduce the need for oxygen and might prevent pulmonary complications.

Therefore, in our opinion there is one group in which it is not necessary to operate, a second group where operation is necessary and beneficial, and a third group where, under present circumstances, pulmonary complications make operation a grave danger.

CHAIRMAN BLALOCK. Have you any comments on results of operation?

DR. MATHEY: We have operated on 5 patients with coarctions in 20 months. One child developed pulmonary edema and died 24 hours after operation. Of the 4 who had beneficial postoperative effects, 1 showed progressive hypertrophy and died for the same reason as those who were not operated. The 3 remaining showed striking clinical improvement and none of these children has shown complications since the operation.

The growth curve of these children is normal. We are not sure of the long-range results of these operations. We are not sure as to the growth of the anastomosis and we do not know whether this anastomosis on an infant of a few months age will give sufficient caliber of the aorta in the adolescent or in the adult.

In the 11 cases we have observed, 5 at operation and 6 others at autopsy, none required a graft. The anastomosis was very short. It may be easier in the young child to mobilize the two aortic segments and make them meet. We have never observed infantile stenosis of a few centimenters in length.

DR. GROSS: In dealing with patients of all ages, we have found that those in the first decade of life rarely need a graft. We have used them in only about 5 per cent of the cases in that age group. In the second decade, grafts have not been necessary very often—8 or 9 per cent. But in patients beyond 20 years of age, we have used them in about 20 per cent of all patients. We feel very hesitant about tackling an adult with

coarctation, unless there is some kind of a graft on hand for use in case of necessity.

CHAIRMAN BLALOCK: In Baltimore, we always have a graft available, but have used grafts in a lower percentage of cases than has Dr. Gross. I have had the feeling, perhaps wrongly, that one should accept a slight reduction in size of the aorta at the site of the anastomosis in preference to using a homograft. After a period of years, this question will be answered with certainty but it seems to me, even though the results thus far with homografts are brilliant, that more time is required to establish their true value.

DR GROSS: I certainly had a few patients in former years where, in attempting primary anastomosis, I did not get as good a pathway as desired and I ended up with some cases feeling that if—appreciating the disadvantages—I had used the graft, a more normal pathway could have been obtained and probably a better result given the patient.

CHAIRMAN BLALOCK Have any of the panel used plastic materials in the treatment of coarctation, where the two ends could not be brought together? Are there any further comments on the use of grafts in the treatment of coarctation?

DR. GROSS: I take a slightly different point of view from that which Dr. Mathey had expressed on coarctation in babies. He indicated that those youngsters under 1 year of age with coarctation who are not operated upon, have fatality rates which are very high, and, indeed, in his series, all the nonoperated ones died. Our experience has been quite the reverse, and I have been very hesitant about operating upon youngsters under 1 year of age for coarctation, fully realizing that such operative procedures have been carried out now by many surgeons in a very effective way.

We have found that babies under 1 year of age are apt to develop left-sided failure. I assume that comes because they have previously had an open ductus which relieved the hypertension in the upper part of the body and then, as the ductus closed at three or four months of age, they suddenly have a left ventricle working against a circulatory bed which is not big

enough to receive all this blood. We have found that if those children are subjected to a good medical regimen, which includes low-salt diet, digitalization, sometimes oxygen and hospitalization, they will usually regain compensation very well, and then go on through childhood in a most satisfactory manner. Of the 13 that we have handled in that way, 12 survived. I am a surgeon and talking against the use of surgery here. We prefer to handle these babies in that way, and then carry them along to 11 or 12 years of age, when an anastomosis in the larger aorta is more apt to carry them through the adult years with a better pathway. Rightly or wrongly, that is our policy for the moment.

DR. MATHEY: One of the first things we did was to study the latest work of Dr. Gross, and we noted that his prognoses were very different from ours because, as he has just told you, he was able to arrive at stabilization of the symptoms by medical aid and to operate at a later stage. That is why, during our first cases, we tried to treat these children medically, and although we were able to see stabilization in a few cases, we saw aggravation in others.

I cannot explain the divergence between Dr. Gross's statistics and ours. I believe we must remember that we can never operate immediately on the simple basis of coarctation and hypertrophy in a child. We have also observed, as has Dr. Gross, that certain acute phenomena at the time of the closing of the ductus arteriosus do not always indicate a dividing line between children who could be operated on and those who are able to stabilize and wait for a later operation.

In other cases—for us, the majority—the question was quite different. For example, a child was operated eight months after diagnosis of coarctation, because the x-rays showed a progressive increase in heart size and the electrocardiograms revealed progressive left ventricular hypertrophy.

I believe that one cannot decide the necessity of surgery by a single examination. This requires evidence of progressive symptoms in spite of good medical treatment. However, one must not wait too long, because the development of pulmonary complications may make operation difficult and even dangerous.

CHAIRMAN BLALOCK: Dr. Robles, what is the preferred age for the surgical treatment of coarctation?

DR. CLEMENTE ROBLES: We are convinced that surgery in coarctation of the aorta should be performed on young people. Therefore we prefer to perform the operation on children, preferably before the age of 10 years. However, there are times when the diagnosis has not been established, and the surgeon may take patients over 10 years. We believe that patients more than 22 years old should not be operated on, or at least that patients over this age carry a heavy operation risk. We have no experience with operations on children under 1 year of age.

CHAIRMAN BLALOCK: Dr. Gross, what age do you prefer?

DR. GROSS: We have generally tried to stall off operation on youngsters with coarctation, but in some instances, because of the presence of failure, cerebral hemorrhage, and so forth, we have operated on youngsters 4 or 5 years of age.

In general, if everything is going along reasonably well, we like to delay operation until the age of 10 or 12, feeling that at that time operation is relatively simple, and that we can establish an aortic pathway that will be satisfactory in adult years.

CHAIRMAN BLALOCK: Dr. Robles, should a child of 10, with moderate hypertension and no other symptoms referable to coarctation, be operated on at that age or should the operation be deferred until a later age?

DR. ROBLES: We believe that he should be operated on then because, generally, at that age, the symptoms are not very marked but the coarctation is present and very clear We remember some patients in whom there were doubts on the part of the clinicians as to whether an operation should be effected because these symptoms were slight, that is, there was only slight hypertension. Nevertheless, the operation was carried out and, to our great surprise, we found that the aorta had a complete coarctation with an extremely narrow diaphragm which completely blocked circulation. The children should not be kept waiting when the symptoms very clearly indicate an operation.

We believe that an operation should not be effected later, because, later on, when the symptoms are clear, the walls of the

aorta may be slightly altered, and the operation is much more difficult, or even impossible.

PATENT DUCTUS ARTERIOSUS

CHAIRMAN BLALOCK: What is the preferred age, in your opinion, for the surgical treatment of patent ductus arteriosus?

DR. ROBLES: It has been said that the best age is at 3 years and the next best is between 3 and 10. I believe that this clinical *criterion should be changed, and that it basically depends on* whether or not complications exist. If the patient has no complications, then, certainly, the operation may be done with more ease at 3 years of age, but if there are complications, the operation should be carried out as soon as possible.

This means that some patients with slight signs of left hypertrophy or danger of persistent pulmonary hypertension should be carried to the operating table as soon as possible This criterion has caused a modification of the classic concept of the age at which the patient should be operated, and there is an increased tendency to operate on children under the age of 3 years.

We already have some experience in these children and we believe that the operation can be done under these conditions with safety. The basic problem is anesthesia. Actually, anesthesiology has sufficient resources to solve the problem of anesthesia in small children Therefore, it has been stated that the best age is 3 years. At present there is a tendency to operate as soon as the diagnosis is established, regardless of the age of the child.

PURE PULMONIC STENOSIS

CHAIRMAN BLALOCK: Sir Russell Brock, should a child of 10, with valvular pulmonic stenosis, without much enlargement, and with a right ventricular pressure of 90 mm., be operated upon or should operation be deferred?

SIR RUSSELL: Obviously, one cannot give a straight, unqualified answer to this.

This matter was discussed to a certain extent in the panel on

congenital heart disease on Monday, and the principle expressed there was that it would be quite wrong to operate routinely on all patients with valvular pulmonic stenosis, and I am fully in agreement with that opinion. There are plenty of patients with valvular pulmonic stenosis in whom there would be no justification for operation.

Of course this question has been very cleverly framed, because virtually a borderline case is being presented, and I think the answer is that, one cannot judge entirely by this one figure of 90 mm, even if one were to accept it as being accurate on all occasions. It may be that the pressure is only 90 under the conditions of catheterization, and not under conditions of extreme exercise. We don't know what it is going to be during the coming years. And so, I think I would prefer to decide by the other clinical features. For instance, it was not stated whether this patient has evidence of an atrial-septal defect with a right-left shunt. Certainly, if the patient was blue or was intermittently blue, with evidence of shunting from right to left, then, I would not hesitate to advise operation

If the septum was closed, I would be influenced by such things as evidence of raised venous pressure and enlargement of the liver, particularly occurring after exercise. If there was evidence of raised venous pressure, even with a right ventricular pressure of only 90, I would be influenced toward operation I would also pay very great attention to changes demonstrated on the electrocardiogram. If there is any evidence of right ventricular strain, I would certainly advise operation.

I feel that if one is dealing with right ventricular pressure alone, I should think somewhere between 75 and 100 mm. would be what one would take as the dividing line. Therefore, this figure of 90 is borderline.

Dr. Gross· I should like to emphasize what Sir Russell said about the pressure at rest, at which so many catheterizations are done, compared with that at exercise. Some cases with rather slight elevation of pressure in the right ventricle at rest, can show very high pressure when catheterization is performed

during exercise, which gives you an entirely different picture.

CHAIRMAN BLALOCK: The next question I should like to ask Sir Russell is, are you satisfied with the procedure for valvular pulmonic stenosis devised by yourself, or do you think that an attack under direct vision, with temporary stoppage of the circulation, will be the ultimate answer?

SIR RUSSELL: Dr. Blalock, I have already stuck my neck out and said that, in general, one ought to see what one is doing, as for example in the closure of the atrial-septal defect. The question in regard to valvulotomy is whether I ought to see what I am doing or to do it in a less direct fashion. The answer is this: are you going to get a reasonably satisfactory result by a relatively closed method of pulmonary valvulotomy, and what is the mortailty? What are the advantages, both in the result and in improved mortality, with temporary stoppage of the circulation?

Of course, there is very little doubt that in theory, it is better to look at the valve, cut it open, and sew up the pulmonary artery. So far as my own experiences are concerned, the mortality of pulmonary valvulotomy by a closed technic is extremely low. I believe that the correct figures are these: In 90 cases, I have lost 12 patients, but of the first 7, I lost 6. Actually, the majority died before I did the valvulotomy, that is, they died either during induction of anesthesia or before the chest was opened. They were very bad risks. However, of the last 83, I lost 6 patients.

Now, in each of those, there was some very good reason why one would expect to have a bad result. For instance, 2 of them had had anastomosis done and were in chronic right-sided failure, 1 had episodes of paroxysmal ventricular tachycardia, and so on. Our experience is that in the average, uncomplicated case of pulmonary valvular stenosis, the mortality is extremely low. Therefore, I do not think one would improve the mortality by direct observation.

Is the mortality going to be greater with an open division under some form of arrested circulation? I think it would be a

very brave man who would say that, at present, you could do 100 cases with a low mortality with these methods of arrested circulation. It may well be, of course, that when we are thoroughly familiar with the new technics, the mortality will be no higher.

The closed method is technically very simple. It means opening one pleural cavity only and one small incision, as opposed to the opening of both pleural cavities and a much larger incision, if you are going to use a temporary arrest of the circulation. Of course, these considerations would not weigh against a fundamentally more satisfactory operation

Now, we come to the efficacy of the procedure of closed as opposed to open valvotomy. I think there is no doubt at all that there are cases in which it is difficult or impossible to achieve as fully a satisfactory valvotomy as one would like with the closed method, but I think, in part, that may be due to the fact that the surgeon has not got either the instruments or the experience to open the valve widely. Indeed I know some surgeons who use a valvulotome with no efficient method of splitting the valve You can get almost complete pressure conversion in many cases with the closed method. On the other hand, I would not deny that there are cases in which it is not possible to do a valvotomy

I recently operated on a patient aged 31 years, in whom the pressure change was disappointing. In such an instance one would be much happier in doing some form of open operation By the way, as Dr Campbell pointed out when he presented our own experiences with the operation, most of these cases are converted from a high degree of pulmonary stenosis to a moderate degree of pulmonary stenosis, in which we would ordinarily expect that the prognosis is probably satisfactory.

The last point that I should like to discuss is the results of the open valvotomy as at present described. We are, of course, very limited in that the only report that I know is the one published in *Circulation* by Drs. Blount and Swan. One must recognize that they are early pioneer cases and have inevitable disadvantages. However, I was very impressed that

their results were unsatisfactory. Maybe it was due to overenthusiasm in what they did with the valve, but it clearly indicates that there is a temptation to do too much to the valve.

One of the things that has emerged from our long-term studies of the effects of pulmonary valvotomy is that in those in whom the heart is at all enlarged, we get a diminution in size of the heart. As Dr. Campbell pointed out, if the heart is not much enlarged and they have not been active, then the heart may enlarge afterwards, but, in general, the heart decreases in size, and sometimes very strikingly. Moreover, the electrocardiographic changes are very striking.

In the cases reported in *Circulation*, I think I am right in saying that in all cases, the size of the heart increased and it was believed that this was due to pulmonary regurgitation, pulmonary incompetence.

It might be argued that this is not significant. Of course, we do not know much about the long-term effects of pulmonary valve incompetence. There is no evidence that it is not harmful and, from the few cases that we have observed, I would say it is harmful, just as aortic incompetence or mitral incompetence is harmful, and I think these patients in whom the heart is enlarging have a poor prognosis.

As for the electrocardiographic changes, although there is temporary immediate improvement from the complete relief of the stenosis in the cases reported by Dr. Blount and Dr. Swan, I suspect that in a year or two, they will show changes of gross right ventricular strain and possibly severe failure.

Let me say again, I do not think this is an objection to the direct, open operation on the valve It may be that it is just a temporary technical imperfection, and when we have more experience in dividing the valve under direct vision, that will cease to be a criticism Certainly, I myself would be prepared, in cases in which I anticipated or actually found the valve to be rigid and resistant to ordinary valvotomy, to do an open division. But, at the present moment, I would not do it as a routine, because I am quite satisfied with our present results. I am very worried in those cases in which the heart is large. They are very

bad risks. I am very worried that one would have a higher mortality. In the cases that we have done under hypothermia, ventricular fibrillation, cardiac arrest, and other troubles are prone to occur in patients with severe circulatory or myocardial changes. When there is a high degree of valvular stenosis, I am afraid of these difficulties.

CHAIRMAN BLALOCK: I think we would certainly agree with Sir Russell that, save in exceptional instances, the present direct attack, which is partially blind, is yielding good results. I think that in Dr. Taussig's group of cases, of the first 85 or 90, we had 7 deaths, and in 2 of these, there had been a previous anastomosis. It would seem to me that except in the hands of people such as Dr. Swan and Dr. Blount, who are making a special study of it, hypothermia should, perhaps, be reserved for use in patients with conditions in which there is no satisfactory method of treatment at the present time—a transposition of the great vessels, and so on At any rate, we have been very pleased with the Brock procedure.

DR. MATHEY: I would like to ask Sir Russell a question At present, we believe, as he does, that in many cases the usual method is the safest, but does he not believe that even now, there are indications for a direct attack with hypothermia in the cases of poor results from previous valvotomy regardless of who did the surgery.

SIR RUSSELL: Yes, I certainly think that if valvotomy has been done and there is evidence of an unsatisfactory relief of the stenosis, then I would certainly like to do a direct open valvotomy, whether I or another surgeon had done the operation.

TETRALOGY OF FALLOT

CHAIRMAN BLALOCK: Sir Russell, are your results with direct attack for the tetralogy of Fallot better than those with a shunting procedure?

SIR RUSSELL: The simplest way to answer this question is in one word: No. I would not like to say that the results are better. I think there are certain features which are more satisfactory, but the mortality is not lower. On the other hand, it is no higher.

Are the clinical results better? I think not, though they are comparable. There are certain aspects of the clinical results, though, that are more encouraging. one can get an improved arterial saturation, and there is less tendency to cyanosis with exercise, whereas cyanosis may occur on exercise with the shunt operations, unless the anastomosis is very large, perhaps too large.

I would, however, like to qualify those remarks. First of all, it has been stated and still is stated that the mortality is high. It has been stated that that is the main reason why the operation should not be performed. I have also heard it said that the operation is impossibly dangerous. Those statements simply are not true.

Out of 120 cases, my mortality was 12.5 per cent. I do not think you can call that an indication of an impossibly dangerous operation. We have been given figures for a mortality twice as high as that in the shunt procedures. I do not say the mortality is any less, but I certainly think there is evidence that it is no more.

The trouble is that you cannot take a violin and play beautiful music without practicing, and you cannot remove a stenosis without practicing. A lot of people imagine that they can pick up an instrument and do this direct operation without practicing, or without difficulty. I think, if you are going to do the direct operation, you have got to realize that it requires a certain amount of intelligent application of certain principles.

One of the great merits, I think, of the direct operation is that it leads the way to evolution of a more normal heart. There are unsatisfactory features with the shunt procedures and there are unsatisfactory features with the direct operations, such as increase in size of the heart, and in some cases an increase in evidence of right ventricular strain. These are almost certainly due to the persistence of the ventricular septal defect. The way to guard against them would be to close the ventricular septal defect, and I think that is the next logical evolution of the surgery of the tetralogy of Fallot.

I believe, when we come to a meeting like this, we see sur-

geons from all over the world concerned with improving intracardiac surgery, operating on the interior of the heart, and trying to cure the lesions within it. There can be little doubt that we have not yet achieved this with the tetralogy of Fallot If we want to do it, the way to do it is to open the heart, remove the stenosis, and sew up the hole in the heart directly, and I hope that is what the direct operations will enable us to do.

CHAIRMAN BLALOCK: I agree with Sir Russell. I do not think that the shunting procedures by any means are perfect. I do not think that the direct attack as used thus far is by any means perfect. As a matter of fact, I doubt if a perfect procedure will ever be devised for all cases. I have seen a good many with so much overriding of the aorta that it is difficult to believe that the heart can be made a perfect one. I think that in future years, there will be some patients in whom shunting procedures are used, there will be others in whom the direct attack is used, as practiced at the present time by Dr. Brock, and still others for whom more perfect methods will be evolved.

There is a good deal of difference in the type of patient that is seen in different clinics. For example, in a dogmatic discussion of the choice of procedure, one is apt to overlook the question of age. Sir Russell, I wonder if you would say a word about the use of the direct attack in infants with tetralogy?

SIR RUSSELL: I think that is a very important question and, actually, I cannot answer it from experience because my experience of operating on infants with the tetralogy of Fallot is insignificant. I have always realized that these children constitute a very grave problem and, frankly, I have been quite glad not to have been faced with a large number of them.

I think the cardiologist with whom I work felt that we could do better by concentrating our resources on the older patients, and these younger children have not been operated on except very rarely, when they have been so gravely ill that they were in quite obvious danger of dying if something was not done to save them. Even those numbers have been small.

In general, I would be more likely to use the shunt operation

on these infants. I am not sure because I have not done a sufficiently large number. I imagine that one of the reasons why these little children are so ill is that they have a more severe degree of pulmonary stenosis; there is greater suppression, both relatively, and absolute, of the pulmonary outflow tract. Their outflow tracts are very small, and consequently more difficult to operate on by direct operation. Particularly, if one did a resection, one would have to consider secondary edema and local bruising, which might well cause even an increased obstruction to such a small child. On the whole, I should think that one would do better with these with an anastomotic operation.

I should like now to consider the other extreme; that is, the very old patients. Originally, I had felt that patients with the tetralogy of Fallot in the early twenties were not good candidates for direct operation and I preferred to do the shunt with them. Lately, I have got just as good results and quite a low mortality even in the twenties. But I would be more inclined to do a shunt than a direct operation on a patient in the thirties, because I think their myocardium has suffered a great deal from banging against the obstruction for a quarter or a third of a century, and, moreover, there is no advantage in the ultimate object of the direct operation, which includes allowing the outflow tract to develop, if the operation is done in a child.

CHAIRMAN BLALOCK: Perhaps my American colleague, Dr. Taussig, is quite surgically minded. At any rate, her position about infants with the tetralogy of Fallot is roughly this: that if an infant is doing poorly, if there is, according to her judgment, a less than 50 per cent chance of this child living to the age of 1 or 2, operation is advised, regardless of how sick he is. We have had some patients brought to the operating room in an unconscious state. It is very rare for a patient with pulmonic stenosis to die on Dr Taussig's service. They usually die on my service.

In these infants, most often, we use the procedure described by Potts, Gibson, and Smith, or, at least, we do that operation in those instances in which the subclavian vessels are quite small.

CONTROLLED CROSS CIRCULATION

CHAIRMAN BLALOCK: Dr. Lillehei, I have several questions regarding controlled cross circulation. The first of these is in regard to the controlled cross-circulation technic. Will you give a few of the technical details on the connection of the two circulations, with particular regard to methods of canalization and the amount of blood pumped during complete cardiac bypass.

DR. LILLEHEI: Controlled cross circulation has provided the surgeon with a method, relatively simple, for open intracardiac surgery, in any chamber of the heart, for as long a period of time as is necessary to correct the pathology present.

The technic is relatively simple, and can be applied by any surgeon who is experienced in vascular surgery. It is even possible that its simplicity may to some extent be its Achilles heel. The use of this technic imposes upon its applicants certain responsibilities. Indeed the failure of surgeons to subject themselves to the disciplines of some hours in the experimental laboratory, thereby familiarizing themselves with the mechanics of the method, will inevitably lead to tragic aftermaths for both the donors and the patients.

The only special equipment needed is a relatively simple pump, which may be purchased for $170. The circulations are linked together by two limbs of plastic tubing. In the donor, the catheters are inserted through a short incision in the groin. One catheter is placed in the superficial femoral artery, so that the tip lies in the abdominal aorta. The position of the tip of the catheter is important for there must be no obstruction to the ingress of blood. The other catheter in the groin is inserted into the saphenous vein, as that the vein can be easily tied off, if it is necessary to repair it. The transverse arteriotomy in the superficial femoral artery is always repaired.

In the patient, the vena cava catheter is inserted so that it draws blood from both the superior and the inferior venae cavae. The arterial catheter is inserted through one of the branches of the aorta, either the right or the left subclavian artery.

The importance of the simple pump is control. For every ounce of perfectly adjusted arterial blood that comes from the donor to the patient, an exactly equal amount of venous blood circulates from the patient to the donor. The blood, in its extracorporeal circuit, never leaves the short length of rubber tubing in the pump, so that problems of sterilization are not present. The tubing is disposed of after each perfusion.

We use very little heparin in the experimental laboratory. Indeed heparin is not necessary. I continue to use it clinically for several reasons. One, it does not give us any trouble. As a matter of fact, after the first few cases we forgot to give the protamine, and no difficulty ensued. So in the last 6 or 8 cases, we have not thought it necessary to give the protamine. Protamine itself has certain undesirable effects. It is better to avoid it. The dose of heparin we have used is 0 75 mg. per pound, both to the donor and to the patient.

I might mention that although the surgery on the donor is minimal, he is anesthetized for two reasons. First, I think he would prefer to be. Second, he has to be; otherwise, the patient would wake up when the donor's blood circulates through the patient's body. I do not think it makes any difference what anesthetic agent you use. We use pentothal curare in most, nitrous oxide in some. The donor, of course, has an intratracheal tube, so you are assured of a good airway.

CHAIRMAN BLALOCK: What risk is there to the donor?

DR. LILLEHEI: We believe it is very small. It has been very small in the experimental laboratory, and, all rumors to the contrary, we have lost no donors. Some 15 human operations have now been performed. We have reserved this technic for those lesions for which there is no other corrective procedure. By "corrective procedure," I mean one that restores the circulation to normal by correcting the anatomic defect or defects present. At the present time, we have done four types of lesions, which I will mention in a moment.

To return to the donor, and the potential risks; they are as follows: First, the problem of disproportion. That is the reason any attempt to use this technic without a pump cannot be dis-

couraged too thoroughly. Connecting up two patients in this manner without a pump would be extremely hazardous. The pump is important for control

If the catheters are not properly inserted—and this is where the laboratory experience is extremely important—they may not draw equal amounts of blood. When we first did this in the laboratory, we often put the tip of the arterial catheter just in the femoral artery. Under certain circumstances, the wall of the artery may be sucked against the tip of the catheter and the blood then fail to enter it. That, of course, is disastrous. If the venous circuit is all right, it will bleed out the patient. The opposite may happen, if the catheters are not properly placed. The vena cava catheter may not draw, while the arterial catheter continues to draw. In other words, proportions are important and disproportions may lead to trouble if they are not avoided.

The second possibility of danger to the donor is related to the first, namely, air embolism If one of these catheters fails to draw properly, air may be sucked through one of the joints. The pump is an extremely effective instrument and will exert two atmospheres of pressure, either negative or positive. Most of these joints won't resist that. Indeed there is no point in building them to resist, because if there is a disproportion, you are in trouble, and you might as well know about it by seeing some air bubbles.

The third possible source of risk to the donor is, perhaps, the one that comes first to your mind, but it is of least importance. blood incompatability. We cross and type these patients very carefully. We type the AVO system. That, of course, must be identical. Most parents will have the same AVO system On occasions, we have not been able to use the parents. In those instances we have used aunts, uncles, grandfathers, and, in one case, a volunteer. Within the next few weeks, we will plan an operation in which an older brother wishes to serve as the donor

Subtypes of the blood are very important We check all the Rh factors, the CDE, large and small, also, the Cal-Duffy, and, in addition, do an indirect Coombs test. That sounds like a lot of typing but, actually, this is routine for transfusion in our

hospital. We have a most excellent blood bank at the University of Minnesota under the direction of Dr. Nullziger. Since he came, two years ago, there has not been a fatal transfusion reaction in our hospital. You may say we do not recognize them. I think the reason is that Dr. Nullziger uses a careful type of crossmatching, as I have indicated, plus an indirect Coombs test on every patient. I believe that all hospitals will adopt this technic in the next few years, because it is entirely possible to have a fatal hemolytic transfusion reaction not picked up by the routine cross match, but which would have been picked up by the indirect Coombs, which takes only five minutes.

The amount of blood we have pumped has been substantially reduced below the basal cardiac output, which is based upon the patient's weight. The amount of blood we pumped is based on the azygos flow experimental studies. "Azygos" has nothing to do with the way we cannulate it, but is related to the experimental studies in our laboratory, and by Andressen and Watson in England. This is, I think, the key to this whole problem, whether you use this method or a heart-lung preparation, or any other type of method in which you circulate blood.

Studies demonstrated unequivocally that the dog will survive with all organs intact, perfectly safely, for a half hour, an hour, even an hour and a half, on only the amount of blood that enters the heart through the azygous vein. That was found quite by accident. That amount of blood was found to be almost fantastically small. This led to the challenge of the heretofore almost universally accepted concept that in a total cardiac bypass, you would of necessity, have to pump the full basal cardiac output. That is not true for dogs, and I am happy to say, it is not true for humans. As a matter of fact, it is further from the truth for humans than it is for dogs. In other words, humans require, relatively, even less blood than dogs. I think it is related to the size of their cardiovascular system in relation to body weight.

The basal cardiac output for most humans is somewhere between 100 and 160 cc. per kilogram of body weight per minute. Actually, the azygous flow studies would indicate that in dogs—and the same is true for humans—one tenth of that amount of

blood, or, 8–10 cc. per kilogram a minute, is adequate to maintain an animal for a period of time sufficient to perform any intracardiac procedure that can now be envisaged.

To be on the safe side, in the first few human cases we pumped more. We gradually have been feeling our way down. We started out with 40 cc. of blood per kilogram of body weight per minute. We are now down to 25. We have seen no difficulty whatsoever. We plan to go lower.

The pump is of ample size and will pump enough blood, based on the azygous flow studies, for the smallest baby or the largest adult. It has a tremendous capacity and can be increased by increasing the speed of the speed changer. The motor runs at a constant speed. The speed changer is like a Buick Dynaflow transmission and it can be increased by putting a little larger tubing in the pump

By increasing the diameter of the tubing from 3/16″ to 4/16″, output can be doubled. That is one of the engineering characteristics of any pumping system of this type. One can get very large increases in pump outputs by small increases in diameter of the tubing.

Studies indicate that at reduced amounts of blood flow, there come into play very important compensatory mechanisms, most of which have been well known to physiologists for many years. One of them is the tremendous A-V difference. For example, normally the venous blood returns to the right side of the heart with 70 per cent of the oxygen in it. For the individual with a normal hemoglobin, that means 14 to 15 volumes per cent. When you pump less blood, the tissues can take out every bit of oxygen in the blood. Actually, we do not like to see that happen. We like to see 2 or 3 volumes per cent of oxygen in the venous blood. The increase in the A-V difference is a very important compensatory mechanism.

It has been said that in the total cardiac bypass, any fall in the venous saturation is a dangerous sign. Nothing could be further from the truth. In many of these patients, whom we measure at the end of the pump run, it is 1 or 2 volumes per cent. That

is where we like it to be. If you have an efficient oxygenator, it leaves the donor again with 20 volumes per cent.

There are four types of lesions that have been corrected. The first one that we did attempt, and in which we have had the most experience, was the interventricular septal defects of the isolated type.

What I have said so far concerns largely the method. As I attempted to emphasize, the important element is experience. If you are going to use it, you should work with it in the experimental laboratory first. The only place where you can learn anything of value in the analysis of human intracardiac lesions is the autopsy room. In the museum the specimens are fixed and sclerotic, and are worthless for you. Indeed, they are worse than worthless, because they suggest that you have to use prostheses. The color is deranged. You will not recognize structures. It is, therefore, my opinion that the surgeon should be present at every autopsy on congenital heart disease, and be there before the pathologist, so that he can have a chance to examine the heart and do a repair before the pathologist cuts it apart.

Our surgical approach to an interventricular defect is a long right ventricular cardiotomy. The defect lies up under the tricuspid valve, which is a somewhat difficult place to get at and one of the rasons why I am very skeptical that blind methods can ever be used for this lesion. Second, you have to see precisely where you are going to put your stitches, if you are to achieve closure. Third, you cannot hope to pull those edges together if the heart is full of blood, but when the heart is empty, they fall together.

To convince ourselves that it could be done, we have sutured these in the autopsy room with #6-0 silk, which has a pull of only a half pound or so. Clinically, we use #3-0 to prevent breaking.

CHAIRMAN BLALOCK. Will you tell us briefly, since I know that you are using both hypothermia and the controlled cross circulation in your clinic, what governs your choice of methods?

DR. LILLEHEI: We feel that hypothermia is the method of choice for lesions in the atrium. Second, we want direct vision.

Third, it has been very successful. There have been 19 cases done by Dr. Lewis and Dr. Markle, and there have been only 2 deaths.

Hypothermia, however, is contraindicated in the present state of knowledge for any work in the ventricle. Experimentally, we tried several years ago to combine hypothermia with the pump. It will not work until we know more about hypothermia. The reason is that ventricular fibrillation may occur the minute you get near the interventricular septum or even the ventricular wall. Therefore, the attempts to close these defects have not been successful.

Panel III

Surgical Treatment of Noncongenital Heart Disease

THE DEVELOPMENT OF CARDIAC SURGERY

CHAIRMAN ROBERT E. GROSS: I regret that we do not have here on this panel this morning a representative from Germany, because cardiac surgery can be said to have started in Germany. A German professor went hunting for deer. He saw an animal and shot. He thought he had hit the animal because it staggered and fell, but shortly got up and ran away. The Professor followed the animal, and noticed that the tracks in the snow showed no blood. He was curious as to why there had not been evidence of external bleeding and when he found the deer he performed an autopsy on the spot, and found that there had been damage to the myocardium, with hemopericardium and all the effects of pericardial tamponade. It was those observations that led him to suggest that one might repair wounds of the human heart and, indeed, he was the first to accomplish this, in the following year.

In 1923, Schmidel of Germany was the first to remove portions of a constricted pericardium. During the last two and a half decades, enormous developments have come in many countries in the field of cardiovascular surgery.

Since this Congress is composed largely of cardiologists I think it appropriate to turn it over to a cardiologist. Hence, I am going to ask Dr. Levine to conduct this panel. I have long felt

that Dr. Levine would make a very good surgeon, because he has always had the ability to see the possibilities of surgery in the treatment of human ailments, and yet he has always been one of the most practical men I have known.

DR SAMUEL A. LEVINE: I think it would be worthwhile to spend a few minutes in an historical vein. I would like to tell you how the medical profession felt thirty years ago, when a half-hearted attack was made on the mitral valve by Dr Cutler. The patient survived for four and a half years and appeared to be somewhat better.

I sent a reprint of this to the leader of the cardiologists of the world, Sir James Mackenzie. He was thirty years older than I was, and talked to me like a father to a son or a teacher to a student. He sent back a note saying, "What a silly thing to try to do! Cardiac disability in valvular disease, especially mitral stenosis, is not due to the mechanical defect of the heart. It is heart muscle failure. It doesn't matter very much what the valve is doing."

That was the concept up to thirty years ago. Everything depended upon the heart muscle and the valves were immaterial. In fact, Sir James used to preach, "Throw that stethoscope away. It has a pernicious influence in medicine."

That is the view that the English school—and, I suppose, the American school—had. There was a lot of good and a lot of bad in most of the things we did. Mackenzie did a tremendous lot of good, insisting that symptoms told the story as to whether a person has cardiac disability or not, regardless of a murmur. There were certain by-products that were harmful in that teaching: the disregard of murmurs entirely and of purely mechanical disabilities that were going on in the heart. In those days we did not have blood banks and antibiotics and the approach was wrong, as we now know, in attacking the mitral valve through the ventricle. Fatalities occurred one after the other and after a year or two it was discarded. Now the whole thing has been changed, the approach coming from the auricle, together with all these other things that have brought cardiac surgery right to the forefront.

New vistas have been opened by this morning's lecture. We are all thrilled. The whole problem, which appeared to be at a standstill in the early days of my work, is wide open, and it is a great thrill for those of us who were at work thirty years ago to see what has happened in the past ten or thirteen years. We can be proud of the advances that have taken place and are inevitably going to take place.

DR. J. K. MADDOX: In *The Lancet* for February 3, 1902, will be found a letter from Sir Lauder Brunton, who was then a physician at St. Bartholomew's Hospital. Sir Lauder Brunton was a very busy man, and he was one of that phalanx of medical moguls who had invaded London from the north and established himself equally as firmly there as he had in Scotland, when he first described the benefits of nitroglycerine. In this letter, he says that he had long been concerned with the problem of mitral stenosis, and the shocking terminal stages of that disease when it resisted medical treatment; so that he had got a license from the government to practice cutting the mitral valve in cats. He had found that the best knife was a ladies' bobby pin, sharpened at one end, which he plunged into the ventricle and made a slash at the mitral valve.

However, in this letter he says, "The first question that arises is whether the mitral orifice should be enlarged by elongating the natural opening or whether the valves should be cut through at their middle, at right angles to their normal opening. I think there can be little doubt that the former would be the better plan, but the latter is more easily performed, and it might be sufficient to effect the desired purpose of facilitating the flow of blood from the auricle to the ventricle." And then he goes on to say that he had been very busy and he had not been able to pursue these experiments as far as he wished.

This letter, three weeks later, formed the basis of a leading article by the editor of *The Lancet*—and *The Lancet* at that time had a tremendous influence in directing medical opinion. Sir Lauder Brunton was taken very severely to task; in fact, his suggestion was described as "unusual, as unwarranted, as dangerous," and his observation that other people should be en-

couraged to do it was particularly castigated. "The suggestion," the editor says, "challenges criticism in two directions: the difficulty of the operation, the doubts as to its efficacy, even if successfully carried out," and some two pages are given to what is virtually a severe trouncing of Sir Lauder.

Dr. Robert P. Glover: After Sir Lauder Brunton came out with that suggestion, there was a considerable amount of experimental surgery on heart valves for the next ten years. Bernheim at Hopkins did a considerable amount of work, and Cushing and Carrel were also interested, and I have a note here that Dr. Cushing asked Dr. Carrel in 1914 whether he thought it would be possible to clamp the root of a diseased heart and carry out serious operations. Dr. Carrel answered: "With regard to Dr. Cushing's question of the possibility of performing extensive operations on a diseased heart, I can say that it can be solved only by experience. It is sure that if the muscle itself is diseased, we cannot make any operation which involves the clamping of the pedicle of the heart and the stopping of the circulation"

Dr. Levine: Let me quickly enumerate the various procedures that the surgeons have been carrying out to help the heart in one form or another.

There are operations being performed for coronary artery disease, for angina in one form or another. We must not forget that thyroid cardiacs are still being overlooked, and if we did not employ medical procedures, the old-fashioned subtotal thyroidectomy would still cure a great many thyrocardiacs. The surgeon has done a lot in that field.

A-V fistulas, we know, produce severe heart failure, and are completely reversible if the fistula is properly repaired.

In pericardial constriction there is much that can be done.

Dorsal lumbar sympathectomy, when the operation is effective in lowering the blood pressure, can cause some severely ill cardiacs to become essentially well.

Aneurysms are being operated upon, and we may have a chance to hear from Dr. de Balsac about the inferior vena cava ligation.

Let us start with Dr. Hufnagel, and his experience with aortic valve disease, aortic insufficiency.

AORTIC VALVULAR DISEASE

Dr. Charles A. Hufnagel: I am afraid a great many of our visitors have already been sated with this problem. I should like to lead into the problem of aortic valvular disease by saying a word about the difference between mitral valvular disease and aortic disease.

As you are all fully aware, the mechanical element in mitral valvular disease is a very prominent one. In aortic stenosis, I think this is likewise a very prominent feature. In aortic insufficiency, however, additional factors do come into play. Among these are the concomitant dilatation of late stages of free aortic insufficiency, and the development of serious conduction disturbances, and associated severe coronary insufficiency.

This brings back to mind the criticism of Sir Lauder Brunton's statement with relation to mitral disease. In aortic disease, too, there is a serious element of failure of the myocardium and abnormalities of ventricular conduction, which certainly make the risk in these patients very different to what it is in mitral surgery.

I think—and Dr. Glover, I am sure, agrees with this—that those of us who do a great deal of mitral valvular surgery, find that most patients with mitral disease are fairly readily controllable before operation, and they withstand operation extremely well. The complications which arise as a direct result of operation and in the immediate postoperative period tend to be relatively few in number and, again, controllable.

In our experience with aortic insufficiency, this is not necessarily the case. In contrast to some of the thoughts which have been promulgated in the past, both in the literature and in textbooks, aortic insufficiency is actually as bad as aortic stenosis in terms of sudden death. It is frequently overlooked that sudden death is an extremely common thing in advanced aortic insufficiency.

Also in contrast to what has been frequently taught in the

past, rheumatic heart disease—rheumatic valvulitis—is a common cause of a predominantly aortic insufficiency rather than aortic stenosis alone.

It has been our general experience that although the murmur of aortic stenosis may very frequently accompany severe aortic insufficiency, as demonstrated by physiologic and hemodynamic studies, it is rather uncommon for serious degrees of aortic stenosis to be associated with free, wide-open aortic insufficiency.

Dr. Levine: In our work on aortic stenosis—and I might say that of all the four valves, the aortic valve is, anatomically and pathologically, the hardest valve to do anything with—we have tried to classify aortic stenosis in five stages. Of course, everyone here can think of reasons why this classification is no good, but it is nice to have a mental picture of where the patient is in relation to the course of his disease.

The first stage is represented by the patient who has the murmur of aortic stenosis, but who is completely asymptomatic

The second stage is a rather vague period when the patient suddenly becomes conscious of the fact that he has a heart It is beginning to palpate and he has a little fatigue. Certainly, there is nothing specific in those symptoms that would suggest aortic stenosis, but in the presence of an aortic stenosis murmur, the complaint takes on a good deal more significance.

In the next stage, there is definite decreased cardiac output, expressed cerebrally by dizziness and syncope and myocardially by angina

Those first three stages can go on for 45, 50, or 55 years. We never know when the patient is going to progress to the next stage, so we cannot generalize easily about the length of time of these stages.

However, in Stage 4, the patient begins to have episodes of pulmonary congestion, at first rather readily taken care of but more difficult in each succeeding episode. At this stage our cardiologists say that an average of two years will pass before death.

Obviously, Stage 4 and Stage 5 represent left ventricular fail-

ure. Stage 5 is true congestive failure. The patient probably does not have long to live, if failure is caused by aortic stenosis.

We are actually turning down far more cases than we have done, because most of our cases have come to us in Stages 4 and 5, and I think that clinicians sometimes confuse mitral stenosis and aortic stenosis.

A good many people with mitral stenosis, and bouts of congestive failure, can be salvaged because the cardiac damage from the mechanical strain is behind the valve and there is a good left ventricle in front. But with aortic stenosis, the brunt of the obstructing effect is on the left ventricle, so that by the time that left ventricle fails no surgery of any kind is going to be very helpful, since the left ventricle is the keystone of the heart.

We have a reasonable mortality and a reasonable degree of success in Stage 2 and Stage 3, where the mortality was 9 per cent. Once the left ventricle has failed the mortality rate increases to 40 per cent in Stage 4, and then goes higher. We have given up trying to help patients in Stage 5, and although we put off those in Stage 4, we have to try with some of them. If surgery for aortic stenosis is going to be done, it is going to have to be done far earlier than has previously been the practice. Early operation here is far more important than in mitral stenosis, because the telling effect is on the left ventricle.

People working in tuberculosis sanatoria see a lot of tuberculosis. They do not see many cases of glioma of the brain. Dr. Hufnagel has seen a good many cases of aortic insufficiency because that is what he is working on, and that is what people come to see him for. In an unbiased medical atmosphere, people come who just have heart trouble. They do not select their lesions.

We see a great many cases of aortic stenosis at Brigham Hospital, many more than we do of frank aortic insufficiency, especially the calcific aortic stenosis in older people. I would like to throw out this one word of caution to Dr. Glover and other surgeons. People can have outspoken aortic stenosis, with the mitral valve not involved, and can live forever.

We just finished a review of about 530 cases of aortic stenosis,

well documented, and it is amazing that the cases of pure aortic stenosis have an average age at death almost as good as a normal person—65 years. So, if you see a patient that you can be sure has aortic stenosis, without insufficiency—by that I mean no diastolic murmur and no signs of mitral stenosis—chances are he is going to live for a long time.

When angina, syncope, or congestive failure start the problem is different. A year or two before that, I would not be able to tell whether he is going to crack in one year or two years. A patient of mine, who came in with walking typhoid fever at the the age of 81, was turned down during the Spanish-American War because he had a murmur at the base of his heart. He got in through the back door and went through the campaign all right. Some fifty years later he came in with typhoid fever, and he had been walking from his office in the Back Bay of Boston to midtown, two miles a day. Four years later he still walks two or three miles a day, with the calcific aortic stenosis. That is what some of them can do.

DR. GLOVER: I have a pet armchair theory about calcification of the aortic valve, Dr Levine, and I would like to have you answer this, if you can.

Obviously, most of these patients have rheumatic heart disease. Once the tiny commissures of the aortic valve begin to thicken, there is, at an early stage, obstruction to the outflow of blood from the left ventricle. The aortic leaflet of the mitral valve and the aortic valve itself, is the portion of the outflow tract of the left ventricle that takes the beating from the progressively more strongly working left ventricle. About half of our mitral valves have some calcium on them, and most of the calcium, as a rule, is on the aortic leaflet or over at the posterior or medial commissure of the valve.

Now, is it reasonable to assume that on a background of rheumatic heart disease, trauma from the high flow pressure coming out through the outflow tract—the highest in the body—is a good reason why calcification occurs so much earlier and so extensively and so commonly in this lesion as compared to other places in the heart?

Dr. Levine: I believe that trauma must play an important role. The false thinking that has been going on in the past, specially on the part of pathologists, was that when they found calcification of the mitral valve, it was rheumatic mitral stenosis. When calcification was found in the aortic valve, until recently it was thought to be Monckeberg's sclerosis.

Calcification occurs in all parts of the body, where there is inflammation, constant trauma, or irritation. For instance, we have calcified tubercle and also calcified suprasellar cyst. Calcification can occur for a variety of reasons. There is no reason to assume that because a man is even 80 years old and has calcified aortic stenosis, it is arteriosclerotic. If it is, what was the murmur doing there fifty years before?

Too many of the so-called pure calcified aortic stenotics have a history of rheumatic fever—rheumatic fever is present, and *when it is not present, you can find the ancillary stigmata.* A member of the family had rheumatic fever, or they had a murmur at the age of 25 when they tried to take out life insurance.

I am convinced that most of them are rheumatic. Therefore, it must start with some inflammatory lesion. From then on, trauma, in the course of time, will produce calcium, and I do not think you can tell retrospectively—or maybe you or the pathologists can tell now, but they couldn't twenty years ago. One of the leading pathologists in the country called all these cases rheumatic twenty years ago, and, ten years later, made exactly the opposite statement. What he thought was rheumatic calcific aortic stenosis, he now regards as arteriosclerotic calcification, or Monckeberg's sclerosis I think some inflammation starts, then trauma usually finishes the job.

Dr. Gross: Dr. Hufnagel, in these people with severe aortic insufficiency, you have two problems. One is the marked regurgitation through the valve, with this to-and-fro motion of blood greatly increasing the work of the heart.

The second is that there may be momentary, sudden insufficiency of coronary flow, because as blood goes back from the aorta into the left ventricle, the coronary flow is diminished and may even momentarily stop or be reversed It would seem to

me that the use of a valve, such as you have so nicely developed, could help the first problem a great deal. While you cannot stop all the back flow, as you have said, you can stop a good deal of it, and probably thereby save the ventricle somewhat. It would seem to have no effect in preventing the catastrophes which come in the second group, from the coronary insufficiencies.

Dr. Hufnagel: We are not entirely sure what is the actual effect of operation upon a badly diseased and greatly hypertrophied myocardium. Nevertheless, the second objection that you raised is a valid one. I believe that operation does not particularly increase the coronory flow. There is, however, another factor which enters into this to a large degree: the total amount of flow necessary for the performance of cardiac work in maintaining a normal output. Certainly, the work load is tremendously decreased, and, once this is so even though the total flow is not increased absolutely, it is relatively.

Dr. Gross: The demands are less on the coronary system.

Dr. Hufnagel: The demands are less. On the other hand, there is another factor which I think comes into play in certain types of valves, and it is difficult to assay this Sometimes at operation on some of these patients with a greatly dilated left ventricle one can see the ventricle decrease in size, within ten or fifteen minutes, because so much of the load is taken off.

Myocardial hypertrophy, obviously, does not change the ventricular muscle. It is not affected in the least in terms of volume or size But ventricular size is affected in terms of total volume of fluid in it, and one can see the tight, tense pericardium become flaccid and freely movable, so the total volume in the left ventricle sometimes must change by 50 to 150 cc very quickly.

Of course, in that sense, too, myocardial contraction is improved by shortening of the fibers.

Dr. Levine: I am not at all sure that aortic insufficiency plays any important role in the production of angina. Angina was present a little more frequently in patients who had aortic stenosis without a diastolic murmur than in those with one. Patients who have luetic aortic disease, aortic insufficiency,

and open coronary ostia may have heart failure but it isn't common to see angina. The question of low diastolic pressure producing angina needs reconsideration.

Maybe we have done enough with aortic valve disease for the present. Surgical treatment of the aortic valve is still in the early stage. Maybe the open operation that will become available will simplify the problem but at present it is a rather tough one.

MITRAL STENOSIS

DR. HEIM DE BALSAC: The future of mitral commissurotomy is dominated by the problem of a possible recurrence of stenosis, more or less a long time after the operation. Most publications have pointed out the happy anatomic, physiologic, and clinical results of the operation, but, as good as the surgical situation may be, it does not consider the causes. The lesion may continue its progress, and even though the commissurotomy may have separated the valve, the lesion may not have been completely relieved and the stenosis may continue to cause trouble. The disunion is affected by breakage and not by section.

Every doctor is haunted by the possibility of mitral stenosis reappearing after the commissurotomy. Such a reconstitution of mitral stenosis is not very frequent but it does exist.

Two years ago, at the European Congress of Cardiology in London, in September, 1952, we reported 1 case with anatomic-reversion. Since then, several patients treated by commissurotomy, who improved within a certain period of time, later presented clinical and hemodynamic changes that could only be explained by a recurrence of the stenosis.

The seriousness of these troubles led us to try a second operation, at which time the surgeon found a stenosis and performed another commissurotomy. One of our patients was thus reoperated by another surgeon. Similar results have been recorded elsewhere in Europe. We are not the only ones to have such recurrences.

On the basis of the reappearance of clinical aspects, 4 or 5

subjects out of 163 who had commissurotomies of the residual type had a commissurotomy which was efficient for one year.

What was the degree of rheumatic activity in the cases that we have verified anatomically? The patient presented fibrillation with slight anemia and morbidity which seemed to us to demand immediate surgery. Long hospitalization was warranted by signs of rheumatism, and if we did operate, it was because of the danger of repeated vascular accidents. We know from such cases that postoperative lesions could have occurred But the patient reported at the Congress never showed signs of rheumatism before the first or second operations. The other 2 clinical cases show the same lack of rheumatismal phenomena.

In another case, the same condition coincided with pregnancy. General size seems to us to be a constant factor. All our sick people were young adults or adolescents. The youngest one was 12, the others were 19, 23, 25, 26, and 29 years old.

When the "finger fracture" commissurotomy was sufficient—6 of them were operated in the beginning—it was, at least, satisfactory to the surgeon. There was, in our cases, a complete enlargement effected by the dilator, which separated the two commissures, but in this case, a second operation was necessitated very shortly after the intervention. Therefore it does not seem to us that one can consider a recurrence to be the result of an insufficient enlargement of the stenosis.

The facts summarized are not sufficiently numerous or accurate to give us a definite conclusion. Therefore, because of the importance of this question, we thought it wise to raise the problem in this meeting. We believe that during this discussion, we can establish the percentage of stenosis that recurs after commissurotomy and establish those which were supported by clinical and pathologic examination What is the influence of age or of rheumatic disease? How long should be the waiting period, after all manifestations of rheumatism have disappeared, before operation should be attempted, and what therapy should be adopted in order to achieve stabilization before the operation or to prevent a recurrence after commissurotomy?

DR. LEVINE: It is very clear to me that a mitral valve that

has been split or fractured by the surgeon has remained open as long as it appears to be doing right now. When surgery first came into use, it seemed to me that all the surgeon could do would be to set the thermostat back. The rheumatic process that took twenty years to produce a fish-mouth mitral, getting down to .7 square centimeters from 3.5 or 4 square centimeters, was starting all over again, would retrace its steps, because the underlying disease has not been halted by surgery. But it does not seem to be working out that way (of course, there may be some cases that are regrowing and fusing together) and the picture is somewhat similar to pericardial surgery.

The patient develops pericardial constriction and calcification in the course of five years after some inflammatory lesion; the surgeon operates, and most of the patients retain their improvement for a great many years. There has been very little in the way of recurrences. Now, what does the surgeon do that stops whatever started the pathologic process? I don't know.

This problem leads us to the question of pericardial constriction. I should like to say one word from the medical point of view about some cases of mitral stenosis, just to take the edge off the superenthusiasm that might exist in some surgical atmospheres. There are a fair number of cases of mitral stenosis that get along tolerably well for a great many years, and no one should be told that the diagnosis of mitral stenosis is an indication for surgery. Last year, I saw a 71-year-old patient who had mitral stenosis and auricular fibrillation and I checked the case with one of the house doctors, and left the bed. After I left, he turned to the house officer and said, "Is that Dr. Levine?" He said, "Yes." The man said, "Well, I saw him twenty years ago." He was a janitor, walking up and down two or three flights of stairs frequently during the day. When I looked up my records of twenty-one years before, I found I had made the same diagnosis: mitral stenosis, auricular fibrillation, and a calcified valve.

Now, of course, that is one case, but there are others like it around. In general, I think the physician's job is to see which way the wind is blowing. When the patient is doing well

enough, I think it is just as well to let well enough alone. When things start going down or something new is occurring, that may be the time to act.

DR. MADDOX: Dr. Levine, Dr. de Balsac wanted to know, as you probably remember, the answer to one or two specific questions, before you leave the subject of mitral stenosis. He described five, I think it was, cases of reclosures, and he wanted to know what the American experience has been, if they have encountered reclosure, and what they would do about it.

DR. LEVINE: I think Dr. Glover is in a position to answer that. I am not surprised that they closed. I am surprised that more of them didn't close.

DR. GLOVER: I understand that Dr. de Balsac has 5 cases out of 163, in which he feels the recurrence has taken place. I also understand that those 5 were in patients from the age of 12 to 29.

One might bring up the point there that these are somewhat younger than, certainly, the average age of our group. We have done some 65 of these cases and our average age is around 37, and it might well be that from 12 to 29, certainly at 12, they are right in the period of rheumatic fever recurrence or activity, and that after they get a little older, their rheumatic activity is somewhat less. That would seem to be a reasonable suggestion.

I am sure that in the next year or two, there are going to be many reports of recurrence of stenosis. The question arises how the commissurotomy was done, because it is presently being done by very many surgeons (and should be) but some of them are just taking their finger and jabbing it through the valve and quickly retracing their steps, tying off the appendage. That is not a commissurotomy, when you feel it is split a little bit on either side. We feel that you ought to carry this commissurotomy all the way out to the myocardium.

Obviously, you are going to run into some valves that are so badly damaged that you cannot do that. There will, quite possibly, be some of those valves on which you could not do

a complete commissurotomy because of calcification or extensive fibrosis. Maybe some of those will recur. But it should be the pathology of the valve which limits the commissurotomy and not ignorance of the surgeon.

You must also realize that the anterolateral commissure not infrequently lies parallel, for the last centimeter or so, to the myocardium, and when you are splitting with the finger you may split and come out against the wall and not appreciate the fact that there is quite a lot more commissure left. You might even do the same thing with a straight knife. That is why we use a Mook knife, so that you can be sure that you are getting all the way out to the myocardium, for if you leave a good portion of the commissure uncut you will not do a complete commissurotomy. Then when the commissure has been opened, you must advance your finger through the valve and do a subvalvular dissection, because, not infrequently, the chorda tendineae are sort of stuck to each other or stuck to the wall and you must, with gentle palpation, free them to give mobility.

If one is able to do a complete commissurotomy like that, it will be a long time before stenosis recurs. The terminal state will have been pushed 'way back, I am sure. But if you do not do that, then you have not done a commissurotomy, and it may be, as I said, that the pathology will not let you do it.

PERICARDIAL CONSTRICTION

Dr Levine: Let us turn for a while to pericardial constriction. Dr. Maddox has something to tell us.

Dr. Maddox: This interests me very much because of the difficulty I have in making a definitive diagnosis in this condition, and that is where I would like some help I feel that many of the classical signs and symptoms can be mimicked by other diseases, and even the hemodynamic pressure curves from the auricle are not necessarily specific.

I feel, of course, that it is quite essential in the beginning to eliminate, if we can, other types of actual cardiac failure, such as isolated myocarditis, myxoma of the auricle, and also tricuspid lesions, and so on. I think that can be done, usually, in

the advanced case, but what bothers me more is the case with little or no calcium, and very often with some degree of cardiac enlargement. I feel here that the electrocardiogram has a special role, and it stands by one, I think, more than many other of our laboratory tests.

Then, I have found, in conversation with people in Europe that the etiology of tuberculosis is beginning to be doubted. They do not feel that it produces, perhaps, as many cases of constrictive pericarditis as we had thought, and they base that on the fact that communities with the highest incidence of tuberculosis do not necessarily have the highest incidence of constrictive pericarditis, and that very often there is no obvious sign of tuberculosis elsewhere in the body and there is no history of tuberculous pericarditis.

I think we must recognize that this condition, however, has been proven histologically to be tuberculous in at least 50 per cent of cases and that the presence of calcification does more frequently follow tuberculous lesions than in those of other etiology.

The other suggestions as to etiology, such as trauma, direct injury, benign pericarditis, rheumatic pericarditis, and pneumococcal and staphylococcal infections of the pericardium, must, I think, form a quite small proportion of causes.

The problem as I see it, is whether tuberculosis is sufficiently frequent a cause for us to operate upon cases of tuberculous pericarditis at an earlier stage and before they go on to an irreversible (or relatively irreversible) constriction of the pericardium, with intramyocardial fibrous extension? Should we see that the surgeon operates under a cover of streptomycin, and that it should be kept up for a long period afterwards? How extensive should be the resection? Should it be done in one stage over both ventricles. Is it necessary to clear the whole rings of fibrous tissue around the heart or is it enough just to divide these rings when they are found? I should be interested if one of my surgical colleagues here could answer those questions.

DR. SAMUEL LEVINE: The recognition of pericardial constriction in younger people, on the whole, is not quite as difficult as

in older patients, because we do not have to think very much about coronary artery disease. There are some older people, over 50, even over 60, who have pericardial constriction. They can simulate cases of coronary artery disease. There is vague chest discomfort, which comes in coronary artery disease, too. They have abnormal electrocardiograms, and they have cardiac symptoms and hypertension. They have valve disease, they have syphilis, they have thyrotoxicosis. When you start ruling out the various things, you would therefore have to give brief thought to the possibility of pericardial constriction.

There are a few clinical points about this that deserve attention. I regret to say that the whole conference this week has, in a way, thrown old-fashioned clinical medicine out of the window. There are a lot of the new things that we have to know and, very rightly, that has been emphasized, but we should not forget that bedside medicine still has a place in the care of cardiacs.

One thing that you may detect that will give you the first clue that the patient has a pericardial constriction is that the apex of the heart will show a good diastolic rebound upon careful examination. This is a thing for us to look at and to feel. *You may see a patient who has about as much of a rebound* in the apex region in diastole as the average person, and a movement thrust, typical of the average person with a systole in a normal heart. A diastolic rebound of the apex makes you think of pericardial constriction.

A second prosaic observation is seeing the neck veins stick out when the patient is able to lie flat. The venous pressure has been over 200 mm. After orthodox therapy, the venous pressure stays high, and the patient is pretty comfortable lying flat. That should make you think of pericardial constriction.

Then, a third sign, which is worth paying attention to, is a suffusion that appears over the face directly after the patient lies down. A cardiac coming into the clinic may look like other cardiacs. There isn't very much that is particularly noticeable in the face. He lies down, and one minute later his neck and his face are suffused. The veins stick out. He is quite blue. When

that is striking, there are two thoughts that must come to your mind. They both have the same mechanical effect. One is tricuspid stenosis, which is really a pericardial constriction inside the heart; the other is pericardial constriction. The blood can't get through the chambers of the heart very readily, with that change in posture, and the tissues become suffused. The observation has been very helpful to me with these patients

And one last clinical point: Some cases of pericardial constriction are due to trauma. They have had a hemorrhage in the pericardium from an automobile accident. They recover, but the blood is the basis of subsequent calcification. We have two patients of that sort, with pericardial constriction, calcification, and blood in the pleura. One of them had had his chest tapped some years before, when the trauma occurred. Blood was taken out of the chest. He had calcified pleuritis and calcified pericarditis. He did very well with surgery.

DR. GROSS: Dr. Maddox asked about whether the one- or two-stage operation was suggested. I assume from that that in Australia, the two-stage procedures are sometimes employed. In this country there has been a greater tendency since the war years to use very wide exposures, either with the longitudinal external incision or transverse midsternal incision, so that one could get a good view of both sides of the heart and do a complete job in one stage

DR. MADDOX: That is true, Dr. Gross. We have been too conservative in our decortication, but the last few cases we have done have followed your and Dr. Blalock's suggestion of a Murray-Ramos, wider exposure and a more extensive resection of the left ventricle first and then right over on the other side.

DR. ROBERT E. GROSS: I think it ought to be emphasized that there is a wider range of ages of patients with this disease than had formerly been suggested. I have seen a child 6 months of age with acute suppurative tuberculous pericarditis, improved by exploration and appropriate streptomycin therapy, but going on to develop a constrictive pericardium. He was operated upon at 1½ years of age and did very well.

At the other extreme, Dr. Levine sent me a man of 70 years of age, who had a perfectly typical picture. He was waterlogged, with constricted pericarditis, and after appropriate medical therapy in preparation for operation, did extraordinarily well after removal of his pericardium.

DR. LEVINE: That fellow was rejected as too old to be operated on. At the age of 73, when he was worse, he was finally operated on, and as you heard Dr. Gross say, he made the most amazing recovery. He walks miles, and he looks like a young fellow. It took us about three or four months with medical management to get him in shape for surgery. He must have had about $5000 worth of human albumin. He had hypoproteinemia. The medical service, I must say, did a perfectly splendid job to get him ready for surgery, and the surgeons did a perfect job, because he is a fit fellow now at the age of 77 or 78.

DR. HUFNAGEL: I think we really do not know very much about the earlier phases of pericardial disease, and I should like to make a plea to try to find out something about it. Our approach has been that if we believe that a patient has pericardial disease of an unknown nature, we will biopsy the pericardium as an initial step. The vast majority of these prove to be acute tuberculosis, and at the time the biopsy is done, we do a complete pericardectomy under cover of streptomycin and isoniazid or any of the other drugs. We have operated on about 6 or 7 such patients and all have done very well, and have as yet, in the last two years, not shown any signs of constriction.

ANEURYSM OF THE AORTA

DR LEVINE· Let us turn for a minute to a new development in surgery, that is, the attack on aneurysm of the vessels, of the aorta.

DR. GROSS: Aneurysms of the aorta, thoracic and abdominal, are by no means unknown. Those of the chest are very apt to be syphilitic in nature, and those in the abdomen are more apt to be arteriosclerotic.

Attempts in the past have been directed largely either at pro-

ducing thrombosis by the introduction of wire or some such material, or wrapping of the aneurysm by a cellophane plastic material, in the hope of stimulating some fibroplastic reaction to help stop this progress of expansion. I think that those are today almost completely replaced by newer technics of removal of portions of the aneurysm, and also, under some circumstances, by completely excising the aneurysm and inserting a graft to re-establish a pathway.

The only comment I should like to make here would be to give a little idea of what has happened to some patients who have had grafts of the aorta, other than for aneurysms, but at least it gives us a background for the use of grafts in these cases who do have aneurysms of the great vessel.

We have inserted 50 aortic grafts. Three of these patients died in the hospital for causes unrelated to their aortic anomalies or disease. One of them died within 6 months from infection. In 6 of the remaining 46 the graft has been in for between 1 and 2 years. In 1, the graft has been in for from 6 to 7 years. In 5, grafts have been in between 5 and 6 years. In 5, grafts have been in for between 4 and 5 years In 8, grafts have been in for more than 3 years and less than 4, and so on

We have not found formation of an aneurysm detectable by x-ray findings in any of these patients. We find that 4 of these subjects do have some calcification in the grafts as seen by routine follow-up plates from time to time. We have had no evidence of peripheral embolism, that is, the breaking off of any thrombus from the interior of these grafts, going down to the legs or the abdomen.

As we see this picture, there is not any doubt that these pathways are not normal vessels, but, from following these patients for grafts inserted for other reasons, I think it gives us considerable courage in going ahead and using grafts for the replacement of aneurysms of the thoracic and abdominal aorta.

Dr. Hufnagel: We have, in the last three years, essentially abandoned all technics except excision of aneurysms, and this includes fusiform aneurysms of all types, and, more recently, even sacculated aneurysms, where general resection is

possible. This would include aneurysms of the arch, which arise an inch above the sinus of Valsalva; if one has that much room, one can excise aneurysms of the arch in most cases. Obviously, the higher in the arch and the more extensive the involvement of the arch, the greater the risk. Aneurysms of the abdominal aorta and below, however, or even of the descending thoracic aorta, can be excised with relatively small risk, this risk being primarily not technical but being due to the intercurrent disease caused by the age of the patient, coronary artery disease, extensive renal dysfunction, and so on.

It is interesting that abdominal aneurysms form a pattern which is almost completely predictable. The renal arteries will seldom be involved. The aneurysm will almost always arise immediately below the renal arteries, and by doing, ideally, a five-anastomosis graft—the aorta and the full internal and external iliacs—one can remove essentially all the dilated aneurysmal part, even though there may be extensive arteriosclerotic disease elsewhere.

We have used homografts, as Dr. Gross has pointed out, in a considerable number of these aneurysms, but more recently have switched over to purely prosthetic materials, which we have had in place now for almost a year, in a total of 15 patients. These have not all been in place for a full year, but the oldest has been in place for approximately that time. We believe it is a highly satisfactory method and, although it is a tedious procedure, it certainly is well worth it, because aneurysm is as malignant a disease as cancer.

DR LEVINE: Let me say, if I may, one additional word about simple clinical medicine as it applies to this problem.

There is a doctor who never saw a case of A-V aneurysm in his life, but he heard the noise that an A-V aneurysm produces, when he heard me imitate the noise by mouth. Shortly after, he saw a patient who had been hospitalized for one year with advanced heart failure, refractory to therapy, with anasarca, and he heard a murmur, just by listening to the chest with a stethoscope. He did not know where it was coming from. But he stopped to wonder where it was coming from, and he started to

hear it in the axilla, in the neck, and in the back. It was very loud and had a point of maximum intensity in the right lobe. He wrote me about it. I had not seen or heard of anything like that, so I said, "Get an I-V pyelogram done." He did so and found that there was something mischievous going on in the right kidney.

My second thought was, "I don't know what it is, but I would get that kidney out. It must be an A-V aneurysm." I thought it was due to previous pyelitis or nephritis, not knowing any other reason why a woman aged 40, who had had babies, should have an A-V aneurysm of the kidney. Dr. Sossman, who is a very sharp-eyed roentgenologist, looked at the film and said, "No, that is a congenital aneurysm of the renal artery, which has broken through into a vein."

This physician could not get a surgeon to operate for a while, but the patient was doing poorly, and, finally, the kidney came out and, without any medication after that, all heart failure melted away. The pathologic specimens did show a congenital aneurysm of the renal artery, breaking through into a vein, producing an A-V aneurysm.

The lesson is that auscultation is still worth something in the practice of medicine, and that a person who has a rare disease that is curable gets no comfort out of the fact that nobody else has it. When the disease is curable, either medically or surgically, it is our job to find it. There is no great harm done and only a little mental disturbance if we overlook an incurable disease, but we must not overlook curable disease.

Index